1-2008

Physical Activity and Blood Pressure in Childhood: Findings From a Population-Based Study

Sam D. Leary

Andy R. Ness

George Davey Smith

Calum Mattocks

Kevin Deere

See next page for additional authors

Publication Info

Published in *Hypertension*, Volume 51, Issue 1, 2008, pages 92-98.
Leary, S. D., Ness, A. R., Smith, G. D., Mattocks, C., Deere, K., Blair, S. N., & Riddoch, C. (2008). Physical activity and blood pressure in childhood: Findings from a population-based study. *Hypertension, 51*(1), 92-98.
DOI: 10.1161/HYPERTENSIONAHA.107.099051

Author(s)
Sam D. Leary, Andy R. Ness, George Davey Smith, Calum Mattocks, Kevin Deere, Steven N. Blair, and Chris J. Riddoch

Physical Activity and Blood Pressure in Childhood
Findings From a Population-Based Study

Sam D. Leary, Andy R. Ness, George Davey Smith, Calum Mattocks, Kevin Deere, Steven N. Blair, Chris Riddoch

Abstract—The pathological processes associated with development of cardiovascular disease begin early in life. For example, elevated blood pressure (BP) can be seen in childhood and tracks into adulthood. The relationship between physical activity (PA) and BP in adults is well-established, but findings in children have been inconsistent, with few studies measuring PA mechanically. Children aged 11 to 12 years were recruited from the Avon Longitudinal Study of Parents and Children. 5505 had systolic and diastolic BP measurements, plus valid (at least 10 hours for at least 3 days) accelerometer measures of PA; total PA recorded as average counts per minute (cpm) over the period of valid recording, and minutes per day spent in moderate to vigorous PA (MVPA). Data on a number of possible confounders were also available. Small inverse associations were observed; for systolic BP, $\beta=-0.44$ (95% confidence interval -0.59, -0.28) mm Hg per 100 cpm, and $\beta=-0.66$ (95% CI -0.92, -0.39) mm Hg per 15 minutes/d MVPA, adjusting for child's age and gender. After adjustment for potential confounders, associations were weakened but remained. When PA variables were modeled together, associations with total PA were only a little weaker, whereas those with MVPA were substantially reduced; for systolic BP, $\beta=-0.42$ (95% CI -0.71, -0.13) mm Hg per 100 cpm, and $\beta=-0.03$ (95% CI -0.54, 0.48) mm Hg per 15 minutes/d MVPA. In conclusion, higher levels of PA were associated with lower BP, and results suggested that the volume of activity may be more important than the intensity. **(*Hypertension*. 2008;51:92-98.)**

Key Words: blood pressure ■ physical activity ■ children ■ epidemiology ■ ALSPAC

Cardiovascular disease (CVD) is the leading cause of adult death in Western societies, and the pathological processes associated with the development of this disease begin early in life. For example, elevated blood pressure (BP), one of the major adult risk factors for CVD, has been reported in childhood,[1] and BP levels have been shown to track into adulthood.[2] Furthermore, BP measured in late adolescence and early adulthood predicts CVD mortality many decades later.[3] Therefore, it is important to establish the determinants of BP in childhood.

In adults, a number of factors associated with BP levels have been identified; for example, there is a well-established inverse relationship with physical activity (PA).[4] In children and adolescents, several intervention studies have been undertaken to assess the effects of exercise on resting systolic and diastolic blood pressure (SBP and DBP). Kelley et al[5] performed a meta analysis of studies that randomized children to an intervention of at least 8 weeks of exercise; the pooled estimates suggested reductions of 1% (SBP) and 3% (DBP) in those participating in exercise, although the confidence intervals around these estimates were wide. In addition, a number of observational studies from around the world have investigated associations between PA and BP in children and adolescents (eg,[6–9]). Several reported no association, whereas others reported inverse associations, but not always consistently for SBP and DBP in both males and females. However, all except 3 of these studies[10–12] used questionnaire responses, mainly self-reported (or reported by the parent if the child was too young), as their measure of PA. Questionnaires provide a poor measure of PA in children, as childhood activity is sporadic[13,14] and children are less able to recall their activity than adults. In addition, in adults it is well documented that, for example, obese individuals overestimate their PA levels, and it is likely that measurement bias will also apply to children. Mechanical techniques for measuring PA, such as accelerometers, may provide a more accurate measure in children.[15,16]

The objective of this study was to advance knowledge in this area through assessing associations between mechanically measured PA and BP in 11- to 12-year-old children from a large, contemporary cohort; the Avon Longitudinal Study of Parents and Children (ALSPAC), in which detailed data on potential confounders of any associations are available.

Received July 31, 2007; first decision August 15, 2007; revision accepted October 31, 2007.

From the Departments of Oral and Dental Science (S.D.L., A.R.N.) and Social Medicine (G.D.S., C.M., K.D.), University of Bristol, UK; the Arnold School of Public Health (S.N.B.), University of South Carolina; and Sport and Exercise Science, School for Health (C.R.), University of Bath, UK.

Correspondence to Dr S. Leary, Department of Oral and Dental Science, University of Bristol, 24 Tyndall Avenue, Bristol, BS8 1TQ UK. E-mail s.d.leary@bristol.ac.uk

Hypertension **is available at http://hyper.ahajournals.org** **DOI: 10.1161/HYPERTENSIONAHA.107.099051**

Methods

Study Population

ALSPAC is a prospective study which has been described in detail elsewhere[17] and on the study website (www.alspac.bris.ac.uk). Briefly, 14 541 pregnant women living in 1 of 3 Bristol-based health districts in the former County of Avon with an expected delivery date between April 1991 and December 1992 were enrolled in the study. Detailed information has been collected using self-administered questionnaires, data extraction from medical notes, linkage to routine information systems, and at research clinics for the children. Ethical approval for the study was obtained from the ALSPAC Law and Ethics Committee and Local Research Ethics Committees.

Physical Activity

All children who attended the 11-year clinic were asked to wear an MTI Actigraph AM7164 2.2 accelerometer (Actigraph LLC) for 7 days. The Actigraph, which detects movement in the vertical plane recorded as counts, has been validated in both children and adolescents against indirect calorimetry,[18] observational techniques,[19] and energy expenditure measured by doubly labeled water.[20] Children were asked to wear the Actigraph during waking hours, only remove for showering, bathing, or water sports, and to record the times they wore the Actigraph, and time spent each day swimming or cycling; the PA of swimming is not measured by the Actigraph as the instrument will have been removed, and the PA of cycling is not accurately recorded by the Actigraph. Further details are provided in Mattocks et al.[21]

Data from children who had worn the Actigraph for at least 10 hours per day for at least 3 days were included, and the following 2 variables were derived. Total PA was the total volume activity including activities at all intensities, measured as the average counts per minute (cpm) over the period of valid recording; associations were calculated per 100 cpm, the difference observed between males and females in this cohort. Moderate to vigorous PA (MVPA) was the average minutes of moderate to vigorous PA per valid day; associations were calculated per 15 minutes per day of MVPA as current recommendations are that children spend at least 60 minutes per day in MVPA.[22] A value of greater than 3600 cpm was used to define MVPA, based on a calibration study conducted in a subsample of 246 children who were asked to perform a series of everyday activities while wearing an Actigraph and a portable metabolic unit (Cosmed K4b^2, Cosmed).[23] For slow walking (4.4 kph), brisk walking (5.8 kph), and jogging (9.2 kph), the average cpm were 2954, 4175, and 7667, respectively, based on 5-minute periods of each activity. Additionally, in the whole cohort 30 cpm equated to 5 minutes of MVPA for a 10-hour day,[24] and as can be seen from the above, the cut point for MVPA (3600 cpm) lies approximately midway between slow and fast walking.

Blood Pressure

At the 11-year clinic SBP and DBP were measured using a Dinamap 9301 Vital Signs Monitor. The right arm was used for 99.7% of the children, and a small adult cuff (upper-arm circumference less than 23 cm) for 73.1%; an adult cuff was used for larger arm circumferences. Two values were recorded and the mean used for analysis. The season of measurement (summer: June–August, autumn: September–November, winter: December–February, spring: March–May), time of day (categorized into morning or afternoon), room temperature, and state of the child (categorized into silent or not silent) were noted.

Potential Confounders/Mediators

At the 11-year clinic, the child's height was measured using a Harpenden stadiometer (Holtain Ltd), and weight using a Tanita TBF 305 body fat analyzer and weighing scales (Tanita UK Ltd). Body mass index (BMI) was calculated as weight (kilograms) divided by height squared (meters); standard deviation (SD) scores adjusted for age were derived separately for males and females.

The 32-week antenatal questionnaire asked the mother to record her highest education level, which was collapsed into categories (none/CSE [national school exams at age 16] to university degree). She also recorded the occupation of both herself and her partner; the lowest was used to allocate them to social class groups (classes I [professional/managerial] to V [unskilled manual workers]) using the 1991 OPCS classification.[25] At enrolment, the mother was asked if she had ever had high BP. Responses to questions asked at 18 and 32 weeks were used to determine whether the mother had smoked at all during pregnancy. Paternal smoking data were obtained at 18 weeks from the partner if available, otherwise from the mother. The date of the last menstrual period reported at enrollment and the actual date of delivery were used to estimate gestation. Infant gender and birthweight were abstracted from obstetric records or birth notifications. A puberty questionnaire was filled in by the child's care provider which included questions on pubertal stage,[26] based on pubic hair development for males and the most advanced stage for pubic hair and breast development for females. Data were only used if the puberty questionnaire was administered within 16 weeks of the clinic visit.

Statistical Methods

Means and SDs were calculated for continuous variables that were approximately normally distributed, medians and inter-quartile ranges (IQRs) for skewed variables and proportions for categorical variables. Associations between BP and the potential confounders were assessed using linear regression, as were associations between the PA variables and the potential confounders. There was no a priori hypothesis that the associations between PA and BP would be different for males and females. However, as gender differences in the relationship between PA and obesity have been demonstrated in this cohort,[27] the interaction between gender and PA was formally tested when modeling the association between PA and BP.

Associations between each PA and each BP variable were examined after adjusting for age at 11-year clinic and gender (Model 1), plus BP measurement factors (room temperature, time of day, season, state of child; Model 2), plus social and maternal factors (maternal education, social class, maternal hypertension, maternal and paternal smoking; Model 3), plus child's BMI and height (Model 4), plus pubertal status at 11 (Model 5); this modeling strategy was similar to that used in previous publications based on BP data in this cohort.[28 29] After fitting Models 1 to 5 for total PA and MVPA separately, they were refitted with both measures simultaneously to assess independent associations with BP. A Spearman correlation coefficient was calculated to describe the association between total PA and MVPA.

Analyses were repeated restricting to children who did not report swimming, then to children who did not report cycling in the week of measurement; they were also repeated using the second BP measurements only rather than the mean of 2 measurements, as first readings are typically higher than subsequent ones when using oscillometric devices. Model 1 was repeated restricting to those with complete sets of confounder information, to ensure that any change in effect size observed in Models 2 to 5 was attributable to confounding rather than missing data. Model 3 was also fitted including birthweight and gestation, to assess the effect of these intermediate factors. The regression coefficients from Model 1 were adjusted for regression dilution bias.[30] The intraclass correlation coefficients used to make the adjustments were derived from a repeat measures study in a subset of 315 children who wore the Actigraph on up to 3 subsequent occasions over the course of a year.[31] Although these corrections provide more accurate regression coefficients, they may be overestimates as it is assumed that all the observed variation in the PA variables was attributable to measurement error rather than intraindividual variation, which is unlikely to be the case.[32] All analyses were performed using Stata version 9 (StataCorp).

Results

A total of 11 952 children were invited to the 11-year clinic, and 59.9% attended. Of those who attended, 92.5% agreed to wear an Actigraph, and 84.6% of those provided valid activity data. Of those who attended the clinic, 97.6% provided BP data. This allowed 5505 (2614 males and 2891 females) to be used for analysis. The mean (SD) age at clinic attendance was 11.8 (0.2) years. The median (IQR) total PA was 644.7 (527.6, 773.0) cpm for males and 528.6 (444.0, 638.7) cpm for females, and median (IQR) MVPA was 25.4 (15.7, 37.8) minutes/d for males and 15.7 (9.6, 24.5) minutes/d for females. Mean (SD) SBP was 104.8 (9.4) mm Hg for males and 106.0 (9.9) mm Hg for females, and mean (SD) DBP was 58.3 (6.5) mm Hg for males and 59.1 (6.6) mm Hg for females. The current guidelines of at least 60 minutes MVPA per day were met by 2.6% of the children (5.1% of males and 0.5% of females). The mean (SD) BPs were 2.4 (0.8) mm Hg and 1.0 (0.6) mm Hg lower in those who met the guidelines for systolic and diastolic respectively.

The characteristics of the children are shown in Table 1. There were only modest differences in the characteristics of these children compared with those who did not attend the clinic, or those who attended the clinic but did not provide valid data.[21]

All of the potential confounders were associated with SBP, DBP, or both (please see Table S1, available online at http://hyper.ahajournals.org), with the exception of age (the IQR was narrow; 11.6 to 11.9 years, so associations were not expected) and social class. All the potential confounders were associated with total PA, MVPA, or both (Table S2).

When investigating the association between PA and BP, there was no strong statistical evidence for an interaction with gender ($P=0.3$ for MVPA and SBP, $P=0.1$ for MVPA and DBP, $P=0.02$ for total PA and SBP/DBP, all adjusted for age and gender); therefore all models were fitted for males and females together. The associations between each of total PA and MVPA and SBP/DBP (separate models) are shown in the top sections of Table 2a and 2b. There were small inverse associations between PA and BP that were weakened but not removed by adjustment for confounders, for both PA and both BP measures. Even after adjustment for body size, which is potentially a mediator rather than confounder so could be considered overadjustment, there was some statistical evidence of associations between PA and BP. In general, when total PA and MVPA were entered into models simultaneously (Tables 2a and 2b, bottom sections), associations with total PA were only a little weaker, whereas those with MVPA were substantially reduced. The correlation between total PA and MVPA was 0.86, but the variance inflation factors were 1.9 and 1.8, respectively, so although standard errors of the regression coefficients increased, collinearity was not considered a major problem.[33]

Analyses were repeated for children who had not reported swimming in the week PA was recorded (67.5%), and then separately for children who had not reported cycling (74.2%), and the results were generally similar to those presented above (data not shown). Analyses were also repeated using the 2nd BP measurement only, and again results were generally similar to those presented above (data not shown). When Model 1 was repeated restricting to children who had complete confounder information, regression coefficients were a little greater than those presented using all available data (data not shown). To investigate the effects of adjusting for birthweight and gestation, these were included in Model 3, and regression coefficients were essentially unaltered (data not shown). The intraclass correlation coefficients estimated from the calibration study conducted in this population of 0.53 for total PA and 0.45 for MVPA were used to correct the

Table 1. Characteristics of the 5505 Children at Mean Age 11.8 Years

Characteristic	Categories	n	Mean	SD
Room temperature (°C) at BP measurement		5505	22.3	2.3
Child BMI, kg/m^2*		5476	18.3	16.6, 20.8
Child height, cm		5477	150.7	7.2
Birthweight, kg		5168	3.4	0.5
Gestation, weeks		5238	39.4	1.8
		n	%	
Time of BP measurement	Morning	2589	47.0	
	Afternoon	2916	53.0	
Season of BP measurement	Summer	1504	27.3	
	Autumn	1281	23.3	
	Winter	1160	21.1	
	Spring	1560	28.3	
Child silent during BP measurement	Yes	4656	84.7	
	No	844	15.4	
Maternal education	None/CSE	651	12.8	
	Vocational	421	8.3	
	O levels	1811	35.7	
	A levels	1359	26.8	
	Degree	827	16.3	
Social class	V	149	3.1	
	IV	654	13.5	
	III Manual	1294	26.7	
	III Nonmanual	1321	27.3	
	II	1242	25.6	
	I	186	3.8	
Maternal hypertension	No	4310	86.5	
	Yes	675	13.5	
Maternal smoking	No	3747	79.6	
	Yes	961	20.4	
Paternal smoking	No	3425	67.5	
	Yes	1648	32.5	
Pubertal stage	1	685	22.2	
	2	1087	35.2	
	3	819	26.5	
	4	401	13.0	
	5	100	3.2	

*Median and IQR as skewed distribution.

Table 2. Regressions of SBP (2A) and DBP (2B) at Mean Age 11.8 Years on Total PA and MVPA

	Total PA (per 100 cpm)			MVPA (per 15 mins/day)		
	β	95% CI	*P* Value	β	95% CI	*P* Value
2A. SBP, mm Hg						
Separate models						
1	−0.44	−0.59, −0.28	<0.001	−0.66	−0.92, −0.39	<0.001
2	−0.33	−0.48, −0.17	<0.001	−0.58	−0.84, −0.31	<0.001
3	−0.49	−0.66, −0.32	<0.001	−0.76	−1.06, −0.46	<0.001
4	−0.18	−0.34, −0.01	0.04	−0.32	−0.60, −0.04	0.03
5	−0.28	−0.50, −0.06	0.01	−0.56	−0.94, −0.18	0.004
Simultaneous models						
1	−0.42	−0.71, −0.13	0.004	−0.03	−0.54, 0.48	0.9
2	−0.16	−0.45, 0.14	0.3	−0.35	−0.86, 0.17	0.2
3	−0.41	−0.75, −0.07	0.02	−0.16	−0.74, 0.42	0.6
4	−0.05	−0.37, 0.27	0.8	−0.25	−0.80, 0.30	0.4
5	−0.002	−0.43, 0.43	0.99	−0.55	−1.29, 0.19	0.1
2B. SBP, mm Hg						
Separate models						
1	−0.36	−0.46, −0.25	<0.001	−0.50	−0.68, −0.32	<0.001
2	−0.28	−0.38, −0.17	<0.001	−0.43	−0.62, −0.26	<0.001
3	−0.30	−0.41, −0.18	<0.001	−0.43	−0.63, −0.23	<0.001
4	−0.18	−0.30, −0.07	0.002	−0.26	−0.46, −0.06	0.01
5	−0.14	−0.30, 0.01	0.08	−0.16	−0.43, 0.10	0.2
Simultaneous models						
1	−0.40	−0.60, −0.21	<0.001	0.10	−0.24, 0.44	0.6
2	−0.22	−0.42, −0.02	0.03	−0.12	−0.46, 0.23	0.5
3	−0.31	−0.54, −0.08	0.01	0.03	−0.36, 0.42	0.9
4	−0.21	−0.44, 0.029	0.08	0.05	−0.35, 0.44	0.8
5	−0.23	−0.53, 0.07	0.1	0.17	−0.34, 0.69	0.5

Model 1=age at 11-year clinic, gender (n=5505).

Model 2=Model 1+BP measurement factors (room temperature, time of day, season, state of child; n=5500).

Model 3=Model 2+social and maternal factors (maternal education, social class, maternal hypertension, maternal and paternal smoking; n=4324).

Model 4=Model 3+child's BMI, height (n=4305).

Model 5=Model 4+pubertal status at 11 (restricted to data reported within 16 weeks of 11-year clinic; n=2518).

observed associations for regression dilution bias. The regression coefficients from Model 1 increased to −0.82 mm Hg per 100 cpm and −1.47 mm Hg per 15 minutes/d MVPA for SBP, and −0.67 mm Hg per 100 cpm and −1.11 mm Hg per 15 minutes/d MVPA for DBP, using separate models adjusted for age and gender.

Discussion

This study, based on a large, contemporary population of 11- to 12-year-olds, has demonstrated an association between higher levels of PA and lower levels of BP after adjustment for a number of potential confounders, and results suggested that it may be the volume rather than intensity of the activity that is important. It is one of the few studies to use accelerometers and the first to our knowledge to compare the volume versus the intensity of activity. After adjustment for child's age and gender and correction for regression dilution bias, the size of associations observed were −2.0 (SBP) and −1.6 (DBP) mm Hg per IQR change in total PA (240 cpm). If these associations translate into those of similar magnitude in adulthood, this could be of public health significance. For example, McMahon et al's meta analysis demonstrated that a reduction of 5 mm Hg in DBP was associated with a decrease of at least 34% for stroke and 21% coronary heart disease,[34] so the lower levels of BP observed in the current study could lead to substantial reductions in these diseases.

As PA and BP have been linked in adulthood in a number of studies,[4] an association in childhood is highly plausible. A number of direct mechanisms have been proposed for the BP lowering effects of PA, including neurohumeral, vascular, and structural adaptations.[4] Alternatively PA may be related to BP indirectly, through body weight, although in this study, adjustment for current body size weakened but did not remove the relationship between activity and BP. It is possible that visceral fat may play a

role, but in this cohort DXA measured total and trunk fat are highly correlated,[35] suggesting that truncal fat is just a marker for total fat, although this may change as the children become older.

Findings from other observational studies in children and adolescents have been inconsistent. Most based their PA data on questionnaire responses that, as previously mentioned, do not provide an accurate measure of PA in children, and may explain the lack of association seen in many studies. A recent study[36] demonstrated that for every 100 estimated-metabolic-equivalent hours per week of self-reported PA there was a decrease of 1.15 mm Hg of SBP in 9 year olds; this amount of PA would move a child from the 5th to 95th centile of activity. Findings based on mechanical monitoring are not strictly comparable with those based on self report because of limitations such as the inaccuracies in the self report of time, and the application of adult rather than child MET values as the latter do not exist. However, in the current study, moving a child from the 5th to 95th centile of total PA was associated with a much larger decrease in SBP than that observed in the study by Gidding et al; approximately 4.8 mm Hg after adjustment for child's age and gender, and correction for regression dilution bias (2.0 mm Hg if also adjusted for potential confounders/mediators). This could be attributable to a stronger association, or alternatively, children in the current study may have been more active than those in the previously published study. Klesges et al[10] used a Caltrac monitor as part of a multimethod approach to measuring PA, but did not find an association between PA and BP (unadjusted correlations were −0.01 [SBP] and 0.00 [DBP]). However, their study from the US was only based on 137 children who were much younger (3- to 6-year-olds) than those in the current study, and activity was only monitored for 1 day. The other 2 studies that used mechanical measures of PA both based their analyses on Actigraph data from the European Youth Heart Study. Brage et al[11] did not demonstrate an association between PA and BP in 589 9-year-old Danish children ($P=0.4$ for SBP and $P=0.6$ for DBP after adjustment for confounders). However, the findings of the current study support those of Andersen et al[12] who demonstrated a negative correlation between PA and BP in a pooled group of 1732 9- and 15-year-old Danish, Estonian, and Portuguese children after adjusting for age, gender, and country (correlations were −0.10 [SBP] and −0.09 [DBP], $P<0.001$ for both). The Brage et al[11] study was based on a subset of the Andersen et al study,[12] and the main focus of both analyses was to consider associations with clustered CV risk rather than BP per se.

The current study has a number of limitations. Firstly, it is possible that different results would have been obtained if all children whose mothers originally enrolled in the study were able to be included in the analysis. However, only modest differences in characteristics have been demonstrated between those in the current analysis and those who did not attend the clinic or did not provide valid PA data.[21] In addition, findings were similar if the minimally adjusted analysis were restricted to those with complete data on all confounders rather than including any with available data, providing some reassurance that attrition is unlikely to have biased results. Second, the study was cross-sectional so the possibility of reverse causality cannot be ruled out, although it is unlikely that the levels of BP would be high enough to lead to a reduction in PA at this age; only 0.5% of the children had hypertension, based on the definition suggested by Jackson et al[37] (blood pressure above the 98th centile, which is approximately 130/72 mm Hg for 12 year olds, using reference centiles developed by the authors). Another limitation of the cross-sectional nature of the study is that it is difficult to disentangle inter-relationships between PA and BMI. It has been assumed that BMI is on the causal pathway between PA and BP, so models including this variable may represent overadjustment. However, it is possible that the relationship between PA and BMI is the other way round, although even if this were true, there is still statistical evidence of an association, albeit weaker, between PA and BP after adjustment for body size. Third, these data were observational and it is possible that confounding could explain the results, although as PA was only weakly negatively associated with higher social position (data not shown) and the associations with BP were largely unaltered by adjustment for a number of confounding factors, this is unlikely. Fourth, it could be argued that 2 BP measures with an automated oscillometric device are inadequate for characterizing an individual's usual long-term BP. However, BP was measured by a trained observer, associations with PA were observed using these measurements, and similar results were obtained if the first measurement was discarded. Also, although greater measurement error in the outcome will result in lower precision when estimating the exposure-outcome associations, in a study the size of ours this is not a major weakness. Fifth, the question asking the mothers if they had ever had high blood pressure could have been interpreted in a variety of different ways, although this is not a major concern as adjusting for this variable made little difference to the effect estimates. Finally, there are some limitations with the measurement of PA. The data were based on a single measure over a 3- to 7-day period that did not necessarily include a weekend day. However, associations between PA and BP were similar in children who had at least 1 valid weekend day (data not shown). Shorter recording periods may measure usual PA less precisely and therefore may attenuate associations with BP; the intraclass correlation coefficient based on repeat measures over the course of a year has been used to quantify the likely effect of such measurement error. It is also possible that activity was overestimated or underestimated because of the Hawthorne effect, ie, children may change their behavior as they knew they were being studied. Previous work investigating this issue has been published[21] and has shown that the difference in total activity between the first day of recording and the mean of all subsequent days is small (17 cpm).

Perspectives

In this study only 2.6% of children met the guidelines of at least 60 minutes MVPA per day, and the median amount was 20 minutes per day. Therefore, increasing activity levels so that guidelines are met would result in reductions of 1.8 (SBP) and 1.3 (DBP) mm Hg (after adjusting for child's age and gender) or more (if regression dilution bias is accounted for), if the observed associations were causal and linear. The findings of our study suggest that encouraging children to increase their levels of PA may help to reduce their current BP, which is likely to track into adulthood. This, coupled with the tracking of their higher levels PA[38] that should continue into adulthood and therefore associate with lower adult BP, should contribute to a reduction in cardiovascular risk. In addition, there is evidence that PA can have long term effects on BP; for example, in a study of Harvard Alumni, participation of adolescents and young adults in vigorous activity while at college was associated with a reduced risk of hypertension 16 to 50 years later.[39] Further studies based on mechanical measurement of PA are required to confirm the inverse association between PA and BP demonstrated in the current study. In particular, the hypothesis proposed that it is the volume rather than the intensity of activity that should be increased should be investigated in other studies.

Acknowledgments

We are extremely grateful to all the families who took part in this study, the midwives for their help in recruiting them, and the whole ALSPAC team, which includes interviewers, computer and laboratory technicians, clerical workers, research scientists, volunteers, managers, receptionists, and nurses.

Sources of Funding

The UK Medical Research Council, the Wellcome Trust, and the University of Bristol provide core support for ALSPAC. This research was specifically funded by a grant from National Heart, Lung, and Blood Institute (R01 HL071248-01A) and a grant from the Wellcome Trust GR068049MA.

Disclosures

None.

References

1. Luma GB, Spiotta RT. Hypertension in children and adolescents. *Am Fam Physician*. 2006;73:1158–1168.
2. Bao W, Threefoot SA, Srinivasan SR, Berenson GS. Essential hypertension predicted by tracking of elevated blood pressure from childhood to adulthood: the Bogalusa Heart Study. *Am J Hypertens*. 1995;8: 657–665.
3. McCarron P, Davey Smith G, Okasha M, McEwen J. Blood pressure in young adulthood and mortality from cardiovascular disease. *Lancet*. 2000;355:1430–1431.
4. Pescatello LS, Franklin BA, Fagard R, Farquhar WB, Kelley GA, Ray CA. American College of Sports Medicine position stand: Exercise and hypertension. *Med Sci Sports Exerc*. 2004;36:533–553.
5. Kelley GA, Kelley KS, Tran ZV. The effects of exercise on resting blood pressure in children and adolescents: a meta-analysis of randomised controlled trials. *Prev Cardiol*. 2003;6:8–16.
6. Marti B, Vartiainene E. Relation between leisure time exercise and cardiovascular risk factors among 15-year-olds in Eastern Finland. *J Epidemiol Community Health*. 1989;43:228–233.
7. Macintyre S, Watt G, West P, Ecob R. Correlates of blood pressure in 15 year olds in the west of Scotland. *J Epidemiol Community Health*. 1991;45:143–147.
8. Boreham C, Twisk J, van Mechelen W, Savage M, Strian J, Cran G. Relationships between the development of biological risk factors for coronary heart disease and lifestyle parameters during adolescence: The Northern Ireland Young Hearts Project. *Public Health*. 1999; 113:7–12.
9. Bouziotas C, Koutedakis Y, Nevill A, Ageli E, Tsigilis N, Nikolaou A, Nakou A. Greek adolescents, fitness, fatness, fat intake, activity, and coronary heart disease risk. *Arch Dis Child*. 2004;89:41–44.
10. Klesges RC, Haddock CK, Eck luteinizing hormone (LH). A multimethod approach to the measurement of childhood physical activity and its relationship to blood pressure and body weight. *J Pediatr*. 1990;116: 888–893.
11. Brage S, Wedderkopp N, Ekelund U, Franks PW, Wareham NJ, Andersen LB, Froberg K. Features of the metabolic syndrome are associated with objectively measured physical activity and fitness in Danish children: The European Youth Heart Study. *Diabetes Care*. 2004;27:2141–2148.
12. Andersen LB, Harro M, Sardinha LB, Froberg K, Ekelund U, Brage S, Anderssen SA. Physical activity and clustered cardiovascular risk in children: a cross-sectional study (The European Youth Heart Study). *Lancet*. 2006;368:299–304.
13. Armstrong N, Balding J, Gentle P, Kirby B. Patterns of physical activity among 11 to 16 year old British children. *BMJ*. 1990;301: 203–205.
14. Armstrong N, Bray S. Physical activity patterns defined by continuous heart rate monitoring. *Arch Dis Child*. 1991;66:245–247.
15. Trost SG. Objective measurement of physical activity in youth: current issues, future directions. *Exerc Sport Sci Review*. 2000;29:32–36.
16. de Vries SI, Bakker I, Hopman-Rock M, Hirasing RA, van Mechelen W. Clinimetric review of motion sensors in children and adolescents. *J Clin Epidemiol*. 2006;59:670–680.
17. Golding J, Pembrey M, Jones R. The ALSPAC Study Team. ALSPAC - The Avon Longitudinal Study of Parents and Children. I. Study methodology. *Paediat Perinat Epidemiol*. 2001;15:74–87.
18. Melanson EL, Freedson PS. Validity of computer science and applications, inc. (CSA) activity monitor. *Med Sci Sports Exerc*. 1995;27: 934–940.
19. Fairweather SC, Reilly JJ, Grant S, Whittaker A, Paton JY. Using the Computer Science and Applications (CSA) activity monitor in preschool children. *Paediat Exerc Sci*. 1999;11:413–420.
20. Ekelund U, Sjöström M, Yngve A, Poortvliet E, Nilsson A, Froberg K, Wedderkopp N, and Westerterp K. Physical activity assessed by activity monitor and doubly labelled water in children. *Med Sci Sports Exerc*. 2001;33:275–281.
21. Mattocks C, Ness A, Leary S, Tilling K, Blair SN, Shield J, Deere K, Saunders J, Kirkby J, Golding J, Davey Smith G, Wells J, Wareham N, Reilly J, Riddoch C. Use of accelerometers in a large field based study of children: protocols, design issues and effects on precision. *J Phys Act Health*. In press.
22. Strong WB, Malina RM, Blimkie CJR, Daniels SR, Dishman RK, Gutin B, Hergenroeder AC, Must A, Nixon PA, Pivarnik JM, Rowland T, Trost S, Trudeau F. Evidence based physical activity for school-age youth. *J Pediatric*. 2005;146:732–737.
23. Mattocks C, Leary S, Ness S, Deere K, Saunders J, Tilling K, Kirkby J, Blair SN, Riddoch C. Calibration of an accelerometer during free-living activities in children. *Int J Pediatr Obes*. In press.
24. Riddoch C, Mattocks C, Deere K, Saunders J, Kirkby J, Tilling K, Leary S, Blair S, Ness A. Objective measurement of levels and patterns of physical activity. *Arch Dis Child*. 2007;92:963–969.
25. Office of Population Census and Surveys. *Standard Occupational Classification*. London: Her Majesty's Stationary Office, 1991.
26. Tanner JM. Normal growth and techniques of growth assessment. *Clin Endocrinol Metab*. 1986;15:411–451.
27. Ness AR, Leary SD, Mattocks C, Balir SN, Reilly JJ, Wells J, Ingle S, Tilling K, Davey Smith G, Riddoch C. Objectively measured physical activity and fat mass in a large cohort of children. *PLOS Medicine*. 2007;4:e97.
28. Roberts R, Leary S, Davey Smith G, Ness, A, ALSPAC. Study Team. Maternal age in pregnancy and offspring blood pressure in childhood in the Avon Longitudinal Study of Parents and Children (ALSPAC). *J Hum Hypertens*. 2005;19:893–900.

29. Brion MJA, Leary SD, Davey Smith G, Ness AR. Similar associations of maternal and partner smoking during pregnancy suggest offspring blood pressure is not influenced by intrauterine effect. *Hypertension*. 2007;49: 1422–1428.
30. Knuiman MW, Divitin ML, Buzas JS, Fitzgerald PEB. Regression dilution in epidemiological regression analyses. *Ann Epidemiol*. 1998;8: 56–63.
31. Mattocks C, Leary S, Ness A, Deere K, Saunders J, Kirkby J, Blair SN, Tilling K, Riddoch C. Intra-individual variation of objectively measured physical activity in children. *Med Sci Sports Exerc*. 2007; 39:622–629.
32. McMurray RG, Harrell JS, Bangdiwala SI, Hu J. Tracking of physical activity and aerobic power from childhood through adolescence. *Med Sci Sports Exerc*. 2003;35:1914–1922.
33. Armitage P, Berry G. Statistical Methods in Medical Research. Blackwell Science, UK 1994;3rd edition:323.
34. MacMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J, Abbott R, Godwin J, Dyer A, Stamler J. Blood pressure, stroke, and coronary heart disease. Part 1, Prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. *Lancet*. 1990;335:765–774.
35. Toschke AM, Martin RM, von Kries R, Wells J, Smith GD, Ness AR. Infant feeding method and obesity: body mass index and dual-energy X-ray absorptiometry measurements at 9–10 y of age from the Avon Longitudinal Study of Parents and Children (ALSPAC). *Am J Clin Nutr*. 2007;85:1578–1585.
36. Gidding SS, Barton BA, Dorgan JA, Kimm SYS, Kwiterovich PO, Lasser NL, Robson AM, Stevens VJ, Van Horn L, Simons-Morton DG. Higher self-reported physical activity is associated with lower systolic blood pressure: the Dietary Intervention Study in Childhood (DISC). *Pediatrics*. 2006;118:2388–2393.
37. Jackson LV, Thalange NK, Cole TJ. Blood pressure centiles for Great Britain. *Arch Dis Child*. 2007;92:298–303.
38. Malina RM, Bouchard C, Bar-Or O. *Growth, Maturation and Physical Activity*. Champaign, IL: Human Kinetics, 2004.
39. Paffenbarger RS, Wing AL, Hyde RT, Jung DL. Physical activity and incidence of hypertension in college alumni. *Am J Epidemiol*. 1983;117: 245–257.